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Volume 38 | Issue 3

Article 2

1976

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Recommended Citation

Schwartz, K. J. and Glock, R. D. (1976) "Swine Dysentery - A Practitioner Update," *Iowa State University Veterinarian*: Vol. 38 : Iss. 3 , Article 2.

Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol38/iss3/2

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Swine Dysentery -- A Practitioner Update

by
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Swine dysentery is a mucohemorrhagic, exudative disease with lesions confined to the large intestine of pigs. Estimates from a 1976 survey by the Livestock Conservation Institute indicate an 88% increase in incidence of the disease in the United States since a similar 1972 estimate. This approximates a total annual loss of \$64 million to the swine industry.

Swine dysentery as a specific disease was originally described by Whiting, *et al.*, in 1921 (18). At that time a spiral-shaped organism was associated with the disease, but oral administration of mixed cultures of spiral-shaped organisms failed to produce the disease. In 1948, Doyle reported successful reproduction of swine dysentery in conventional pigs by oral inoculation with *Vibrio coli* (1). This report, however, could not consistently be substantiated by other workers. The etiology remained largely unknown and unpursued until the 1960's when a trend toward confinement swine units and an emerging ineffectiveness of chemotherapeutic agents resulted in an increase in incidence of swine dysentery. Renewed research efforts again indicated a primary involvement of a large spirochete. Before 1971, the evidence of spirochete pathogenicity was largely circumstantial due to a failure to propagate the organisms *in vitro*, and consequently the inability to fulfill Koch's postulates.

In 1971, Taylor and Alexander propagated a large anaerobic spirochete in pure culture and successfully induced swine dysentery by oral inoculation of pigs

(16). The pathogenicity of the large spirochete was confirmed by Harris, *et al.*, and further characterized and named *Treponema hyodysenteriae* by the Iowa State workers (5). *Treponema hyodysenteriae* is now accepted as the primary pathogenic agent in swine dysentery, but in germ-free pigs this organism alone will not cause disease. Meyer, *et al.*, has recently shown that germ-free pigs develop lesions similar to swine dysentery when inoculated with pure cultures of *T. hyodysenteriae* in the presence of four anaerobic gram-negative organisms (12). It is uncertain whether *T. hyodysenteriae* alone possesses all determinants necessary for pathogenicity. It is possible that the other organisms contribute pathogenic determinants or provide environmental factors necessary for the expression of pathogenicity by *T. hyodysenteriae*. Of course, other parasitic organisms may potentiate pathogenic effects of *T. hyodysenteriae*.

The isolation and propagation of *T. hyodysenteriae*, once tedious, has now become a rather routine technique for researchers, as well as a diagnostic aid for the practicing veterinarian. *Treponema hyodysenteriae* is a gram negative, oxygen-tolerant, anaerobic spirochete always found in high numbers in the colon and cecum of acutely affected pigs (10). Morphologically, *T. hyodysenteriae* is a loosely coiled, spiral-shaped organism with motility by serpentine motion. The organism is hemolytic, stimulated by hydrogen and best propagated at a temperature of 42°C.

Swine dysentery is most commonly observed in 30 to 170 pound pigs but may also occur in suckling and adult animals.

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Pigs are the only known reservoirs of *T. hyodysenteriae* and may shed virulent *T. hyodysenteriae* in the absence of clinical signs. Transmission of the disease appears to be by ingestion of fecal material from either clinically affected or clinically normal carriers of *T. hyodysenteriae*. Acutely affected animals tend to shed organisms in very high numbers which may lead to rapid transmission and more severe, explosive outbreaks. Obviously, strict sanitation is of great value in limiting swine contact with contaminated feces. The organisms appear quite resistant when in moist environmental conditions; they remain viable for 1 week in feces at room temperature. Also, feces-contaminated fomites such as boots, vehicles, and equipment may transport viable organisms, all of which underscore the need for strict sanitation procedures.

The incubation period of swine dysentery is extremely variable but usually occurs within 10-14 days. Diarrhea is the most consistent clinical sign of swine dysentery, with classical signs including blood and mucus in feces (15). The severe diarrhea ultimately leads to dehydration, acidosis and death of the animal. The gross lesions include hyperemia and edema of the gut wall with severe mucofibrinous to hemorrhagic enteritis diffusely found in the large intestine. Gross and microscopic lesions, as well as other pathological changes, have been well described by Glock, 1971 (2).

Diagnosis of swine dysentery should be attempted only with consideration of other potential causes of diarrhea that involve the large intestine, namely, salmonellosis and trichuriasis. Factors to consider include herd history, clinical signs, gross lesions, microscopic lesions and diagnostic aids. A highly reliable method of diagnosis includes a thorough necropsy of affected animals with the microscopic observation of large numbers of spiral-shaped *T. hyodysenteriae* in colonic scrapings using darkfield examinations of wet mounts or brightfield examinations of smears stained with crystal violet followed by Gram's iodine. Salmonella isolation should be attempted from colonic contents, mesenteric lymph nodes and other viscera to exclude salmonellosis, bearing in mind

that both diseases may occur simultaneously.

It has become increasingly apparent that nonpathogenic forms of *T. hyodysenteriae* do exist in both normal and affected pigs. Obviously, nonpathogenic *T. hyodysenteriae* complicate the diagnosis of acutely infected animals as well as the detection of carrier animals. Presently, the most reliable method of detecting asymptomatic carrier animals and confirming acute cases of swine dysentery involves the culture of rectal swabs or colonic scrapings. Songer, *et al.*, have developed a selective medium of trypticase soy agar with 5% bovine blood containing 400 mcg spectinomycin per milliliter which is incubated at 42°C. in an anaerobic environment (14). Kinyon's method of differentiating pathogenic and nonpathogenic *T. hyodysenteriae* is based on the observation that pathogens are strongly beta hemolytic while non-pathogens are only weakly so (11). It has also been noted that both pathogenic and nonpathogenic *T. hyodysenteriae* may be present concurrently in the same animal.

Diagnosis of acutely infected animals, then, can be confirmed by microscopic observation of *T. hyodysenteriae* and/or culture attempts. Culture techniques will detect some asymptomatic carriers but require more expertise and experience. It is thought that the technique may be useful on a herd basis rather than for screening individual animals. Serological tests, such as Joens' microtiter agglutination (9) or Jenkins' passive hemagglutination test (8), have been shown sensitive and reproducible in detecting antibodies to *T. hyodysenteriae* in convalescent animals following clinical disease but are not useful in detecting asymptomatic carriers. More research is needed to develop a rapid, accurate, and reliable test for carriers.

Successful swine dysentery control must include careful analysis of the management situation and a judicious utilization of chemotherapeutic agents. Management efforts must decrease stress that may be in the form of overcrowding, filth, extremes of temperature, transportation, nutrition, or other disease. It is of major importance to practice strict sanitation procedures. Swine dysentery severity can be directly correlated with the amount of

contaminated feces which are ingested. Poor sanitation increases the probability of transmission of the disease within the herd as well as contributes to clinical severity of the disease within individual animals. Movement of swine, vehicles and equipment between herds and infected pens should be carefully controlled.

Chemotherapeutic agents for swine dysentery, unfortunately, have not yielded consistent results in the elimination of *T. hyodysenteriae* from infected herds. Most drugs licensed in the U.S., if efficacious, serve only to temporarily suppress clinical disease with recurrence of dysentery in 3-6 weeks. Presently, drugs of choice used as feed additives for prevention and follow-up treatment include carbadox, lincomycin, and virginiamycin. Acute infections are best treated with injectable or water medications (see table) (6). At the present time gentamicin, lincomycin, nitroimidazoles, tylosin or arsenic compounds appear to be most efficacious. Practitioners should be cognizant of all regulations and withdrawal times governing drug usage.

Unofficial field reports from veterinarians indicate that prolonged treatment of problem herds with nitroimidazole compounds and possibly

lincomycin may eliminate *T. hyodysenteriae* from the infected herds. Currently, there are no nitroimidazole compounds labeled for use as feed additives for swine; to use them as such is illegal. Nitroimidazoles may be legally prescribed by the veterinarian as a drinking water medication. However, both practitioner and client are then responsible and liable for its usage and withdrawal from market animals. Regardless of the medication program utilized, it may be useless if undertaken without proper management considerations. Initial treatment must be for a long enough period of time, usually at least two weeks. This must be followed by an adequate preventative treatment program. Any medication program must be coupled with providing a cleaner, less stressful environment for animals. Any treatment regimen must be carefully thought out and adapted to the individual producer's situation.

Persistence of swine dysentery within a herd may indicate the need for total depopulation. Economic impact will be the major consideration since production will be lost for an extended period. To increase chances of success, depopulation should be undertaken during the warmer, dryer months of the year. The premises must be

TABLE 28.2—Dosage level, duration of administration, and withdrawal time for various drugs used for the treatment and/or prevention of swine dysentery

| Compound | Treatment | | Preventive Level | Withdrawal Time |
|-------------------|------------------------|--------------|-------------------|-----------------|
| | Level | Duration | | |
| | | (da) | | (da) |
| Bacitracin | 100 g/ton feed | 6 | 50-100 g/ton feed | None |
| Carbadox | 50 g/ton feed | Continuously | 50 g/ton feed | 70 |
| Chlortetracycline | 100-200 g/ton feed | 3-5 | 50-100 g/ton feed | None |
| Dimetridazole* | 0.025% in water | 5 | 100 g/ton feed | ND† |
| Furazolidone | 300 g/ton feed | 14 | 100 g/ton feed | None |
| Gentamicin* | 50 mg/gal | 3-5 | ND | ND |
| Ipronidazole* | 0.005% in water | 7 | 100 g/ton feed | ND |
| Lincomycin | 100 g/ton feed | 21 | 40 g/ton feed | ND |
| Neomycin | 140 g/ton feed | 3-5 | 100 g/ton feed | 20 |
| Oxytetracycline | 100 g/ton feed | 3-5 | 50 g/ton feed | None |
| Ronidazole* | 0.006% in water | 5 | | ND |
| Sodium arsanilate | 4.5 grains/gal water | 5-7 | | 5 |
| | 90 g/ton feed | 5-7 | 90 g/ton feed | 5 |
| Tylosin | 0.25 g/gal water | 5-6 | 100 g/ton feed | None |
| | 200 mg/da (injectable) | 3 | | 4 |
| Virginiamycin | 100 g/ton feed | 14 | 25 g/ton feed | None |

* These compounds were not approved for administration to swine in the United States at the time this table was compiled.

† Not determined.

Table was reprinted from *Diseases of Swine*, 4th ed. by permission of ISU Press.

thoroughly cleaned and disinfected. Survivability of *T. hyodysenteriae* is not precisely known, but the organism is viable in moist feces at least 1 week and may survive in pits and lagoons for an undetermined time (3). Therefore, efforts must be made to eliminate all fecal contamination from the farm. If depopulation is undertaken, one must attempt to obtain uninfected replacement stock. Presently, there are no good detection methods available to the practitioner so one must rely on a complete history of the source herd. Specific pathogen free herds may be given consideration as a source of replacement of breeding stock.

Eradication of swine dysentery is not feasible at this time. The high incidence of the disease, the lack of sensitive, accurate diagnostic techniques, and inadequate understanding of epidemiology and of etiological relationships all suggest that attempts must be made to control the disease rather than eradicate it.

The chronicity of swine dysentery within herds and individual animals was long thought to indicate no stimulation of immune mechanisms. Terpstra, *et al.*, demonstrated circulating antibodies specific for *T. hyodysenteriae* in convalescent pigs (17). Olsen has shown that convalescent pigs exhibit resistance to reinfection with *T. hyodysenteriae* (13). Attempts by Hudson, *et al.*, and Joens to orally immunize pigs with attenuated cultures of *T. hyodysenteriae* have failed (7). However, Glock, *et al.*, have reported that intravenous hyperimmunization of animals with inactivated *T. hyodysenteriae* has provided protection against an oral challenge with a homologous isolate (4). Glock has theorized that circulating antibodies may confer protection by exudation at the site of lesions but does not discount potential for local immune mechanisms to be involved. Presently, there is no practical way to immunize pigs against swine dysentery, but the search continues for an effective vaccination method.

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